tional and 2 earlier Cæsarean sections the perinatal mortality could have been reduced to 31 per 1,000, which would have compared very favourably with the 1963 figure of 38.

A comparison of this short series of cases treated conservatively with a similar group managed much more actively raises the question as to just where the optimum rates of interference lie. There is little doubt that a substantial improvement in the perinatal figures would have followed a very modest move from rigid conservatism, especially in the management of mothers delivered after the forty-second week. The lessons and achievements of conservatism should not be forgotten when attempting to define with greater precision the indications for induction of labour and the necessity to expedite delivery in labour.

Acknowledgments: The facts and figures presented are drawn from the Annual Medical and Clinical Reports of the Simpson Memorial Maternity Pavilion prepared by Dr J C H Dunlop, who has the help of Dr John Thomson in the compilation of the pædiatric section. I acknowledge the help of these two colleagues. In that hospital, perinatal mortality is defined as stillbirths and all neonatal deaths.

REFERENCE
Medical and Clinical Report of the Simpson Memorial Maternity Pavilion of the Royal Infirmary, Edinburgh, 1955 to 1963 inclusive

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No induction of labour is done without what in my opinion appears to be a good reason. The reasons arise mostly from the various causes of placental insufficiency. These are: pre-eclampsia, hypertension, renal disease, postmaturity, diabetes, threatened miscarriage, ante-partum hæmorrhage and the second twin. Cephalopelvic disproportion may or may not be associated with placental insufficiency but even when it is not it constitutes in itself a reason. Remaining indications are rhesus iso-immunization and intra-uterine death of the fœtus. The commonest reason is pre-eclampsia and I have already described my criteria for interference (Lennon 1957). The most controversial reason is postmaturity in which I have believed for some long time and I have seen no reason to alter my opinion over the years.

Those who do not believe in intra-uterine lack of well-being on the part of the fœtus towards term or after it will, of course, not rupture the membranes and will not find meconium staining of the liquor. Indeed, by induction of labour we set up for ourselves a worry and a clinical difficulty that does not exist for those who do not rupture the membranes until the dark liquor is seen through the bulging membranes at the introitus. They, of course, may get a live baby just as I do by induction of labour but they are not concerned that there might have been already some slight damage to the fœtal brain, which may later prevent that child passing the 11-plus examination: that this may be more important than previously thought is not unknown to the television viewer and is a challenge of proof to our pædiatric colleagues.

The following findings, however, may be relevant in this context:

For two five-year periods in Bristol, 1943–8 and 1948–53, the cerebral palsy rate per 1,000 live births was the same, 2·5. In the five-year period 1953–8 the incidence fell to 1·6, and since 1958 has again fallen to 0·9. These figures have been statistically analysed. Analysis shows that the declining incidence of all types of cerebral palsy in Bristol is highly significant (Woods 1963).

These figures have been statistically analysed by Miss E H L Duncan. The analysis shows that the declining incidence of all types of cerebral palsy in Bristol is highly significant (Woods 1963).

I would draw attention to the year 1953. This is precisely the year when we changed to a very much raised induction-rate policy, particularly in pre-eclampsia and postmaturity. Other and many factors must be involved in this improvement but I feel that the advantages gained by inducing labour – earlier management or avoidance of placental insufficiency, better uterine action resulting in quicker labours, fewer traumatic and difficult deliveries (Bainbridge *et al.* 1958) – must have played a part.

In a recent publication on indications for the induction of labour (Donald 1961) I read: 'A study of the last analysed nine months' cases shows that induction was performed in no less than 20 per cent of cases which is higher than most other people's figures, but it should be pointed out that, so far as possible, normal cases are filtered off. No less than 15·5 per cent of all those induced ultimately required delivery by Cæsarean section as against an incidence of 10 per cent of Cæsarean section in the hospital as a whole.' I have, therefore, investigated my figures for Cæsarean section (and oxytocin drip) for the past five years (Table 1).

One of my colleagues was so appalled at my 38% incidence that he went and found out his figures; his were 40%. (Most of my normal cases are filtered off.)

Table 1
Surgical inductions

	1959	*1960*	*1961*	*1962*	*1963*
Percentage of total deliveries	45	37·4	42·1	38·7	38
Percentage of inductions given oxytocin	20·5	24·9	21	25·9	30·46
Perinatal loss (uncorrected)	3·38	2·8	2·9	1·94	2·98
Cæsarean section (per cent of all inductions)	1·4	3·5	2·4	2·9	3·3 (0·5 oxytocin)
Cæsarean section (for failure to go into labour)	0	2	0	3	4
Cæsarean section (for prolapsed cord)	0	0	0	2	0

The Cæsarean section rate in 1963 is 3·3% with an incidence of oxytocin drip in 30·46% of cases. The Cæsarean section rate in drip cases was 0·5%. Mr Theobald accuses me of the use of a pharmacological drip as opposed to his physiological drip but I get better results as regards labour with my higher dosage (Theobald 1959). I did try his dosage for a spell and gave it up. I have seen no complications of a serious nature with the hospital use of the pharmacological drip. One exception is the grand multipara: in her case the drip may cause such good uterine action towards the end of the first stage that there is no intermission for the baby, so that in these cases one has to be careful to cut down the drip to a much lower level. The following points with regard to the use of the drip are important:

(1) If the indication for induction of labour is present and real, the state of the cervix will only interfere with the method employed. In an early-gestation pre-eclamptic it may be necessary to empty the uterus immediately by hysterotomy or Cæsarean section or, if there is more time, to attempt to open up the cervix by preliminary drip therapy prior to rupture of the forewaters.

(2) Where the cervix is long but permits rupture of the forewaters time must be allowed for the taking-up of the cervix, hence my delay of drip therapy for twenty-four hours in cases of pre-eclampsia and of forty-eight hours in cases of postmaturity.

(3) It should be remembered that, induction of labour apart, some women never go into labour or, if they do, only unsatisfactorily and then Cæsarean section is necessary. So there is a failure rate even when left to their own state. Persistence with drip therapy, therefore, in induced cases when there is little or no response should be looked at from this point of view.

In the light of our experience should we not now regard spontaneous early rupture of the membranes as an attempt on the part of Nature to initiate a better and quicker labour?

Two fundamental researches must be undertaken: (1) To determine an easy and quick method of estimating placental sufficiency. In Bristol over the past few years we have explored vaginal cytology in this connexion but by the time changes occur the baby may already have perished. We have found some help from œstriol estimations in the urine but they are difficult and time consuming. In one case where twenty-four-hour urine specimens were being collected for œstriol estimations we realized (after delivery) that a good indicator had been the fall in the amounts of urine passed over three successive days. Further investigation of this may reveal an easier assessment than complicated biochemical tests.

(2) To determine an easy and certain method, preferably medical, for induction of labour at all stages of gestation. Release of the progesterone block of Csapo (1961) by some means may provide the answer. And then release of liquor will be debated for other reasons.

REFERENCES

Bainbridge M N, Nixon W C W & Smyth C N (1958) *J. Obstet. Gynæc., Brit. Emp.* **65**, 189

Csapo A (1961) In: Progesterone and the Defence Mechanism of Pregnancy. Ciba Foundation Study Group No. 9. Ed. G E W Wolstenholme & M Cameron. London; p 3

Donald I (1961) *Practitioner* **186**, 549

Lennon G G (1957) *Proc. R. Soc. Med.* **50**, 793

Theobald G W (1959) *Lancet* i, 59

Woods G E (1963) *Develop. Med. Child Neurol.* **5**, 449

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Results from the Perinatal Mortality Survey of 1958

The Perinatal Mortality Survey of 1958 was carried out by the National Birthday Trust Fund. The Survey was directed by Dr Neville Butler, and the first report was published by Dr Butler and Professor Bonham in 1963.

The results given here are based on the main Survey week which was March 3–9, 1958. A questionnaire was completed by the midwife

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